and itching; 630 (24 percent) had severe generalized life-threatening reactions with unconsciousness, dyspnea or throat swelling; 73 (3 percent) had delayed reactions including hyperglobulinemic thrombocytopenic purpura, bloody diarrhea, nephrotic syndrome, hepatorenal syndrome, and CNS involvement (EEG changes, peripheral neuropathy, polyneuritis, transverse myelitis). Serum sickness reactions have been noted as late as ten days after a sting. Infection at site of sting is common, especially after stings by yellow jackets, wasps or hornets, which unlike the honeybee are scavengers.

The diagnosis is made primarily by history; however, skin testing, although not infallible, is confirmatory and should be performed with titration, increasing concentrations of extracts to determine the concentration to be used in hyposensitization. A refractory period of falsely negative skin tests may exist as long as three weeks after a sting. False positive skin tests are not infrequent. Hyposensitization injections are of value in reducing both local and systemic reactions. Duration of hyposensitization treatment has not been established. However, current thinking is that it should continue for at least five years with the injections being given every one to four months after a maximum dosage has been achieved.

Prompt emergency treatment is very important and persons known to be sensitive to hymenoptera should carry an emergency kit at all times. The stinger and venom sac of a honeybee should be scraped off completely with a fingernail, for it takes 2 to 3 minutes for the sac to empty. Epinephrine is by far the drug of choice, and should be injected both at the site of the sting to delay absorption, and elsewhere for systemic effects. Not quite as effective but more convenient for emergency kits are the inhalers containing epinephrine. Sublingual isoproterenol is no longer recommended, as it may worsen shock from peripheral vasodilation because of its beta-adrenergic effect. Antihistamines are also useful in the initial therapy and steroids may be of value later. A tourniquet should be included in every kit.

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Mites in House Dust

RECENTLY THERE HAVE been many reports concerning the possibility that house dust allergy is caused by pyroglyphid mite sensitivity. Evidence of this association has been based on history, finding the mites in homes, puncture skin tests, leukocyte histamine release, passive transfer and relief of symptoms by hyposensitization with mite extracts.

In contrast, Kawai et al have reported that while leukocytes of some house dust reactors release histamine in the presence of mite extracts, others do not.

In addition, Hosen has warned that insect extracts themselves contain histamine and can produce a wheal by either puncture or intradermal test.

Mites are probably an important cause of house dust allergy but other factors may also be involved. Further studies will be necessary to clarify the situation.

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Association of Air Pollution With Asthma

ASTHMA HAS TRADITIONALLY been associated with airborne pollutants from natural sources such as pollens and mold spores, and symptoms are believed to be mediated by allergic mechanisms. Nonspecific irritants may also produce asthma in

susceptible persons. Chemical air pollutants may produce decreased respiratory flow rates in persons dwelling in communities with air pollution, and in particular in patients with chronic respiratory disease. Admissions to hospital because of asthma are more frequent for residents of the more polluted sections of urban areas.

There are two major forms of air pollution: sulfur dioxide-particulates and photochemical smog. Both forms can cause increased airway resistance and interfere with the clearing mechanism of the bronchi. SO_2 is irritating, and can paralyze ciliary action, stimulate mucus production and cause bronchospasm and edema. Similar effects can be produced by ozone and NO_2 in photochemical smog.

The frequency of admittances to hospital for asthma increase the night of and the day following an increase in air pollution. In Japan, United States military personnel had to be evacuated from the Tokyo-Yokohama area because of pollution-induced asthma which responded poorly to bronchodilators. When petroleum refineries began to operate in Yokkaichi, Japan, there was a pronounced increase in asthma among local residents. In the acute air pollution episode in Donora, Pennsylvania, 87 percent of the asthmatics became ill, compared with 43 percent of the town's general population.

Experiments in guinea pigs indicate chemical air pollutants may contribute to allergic asthma. Ozone, NO_2, and SO_2 enhanced antibody production to nebulized albumin and pre-exposure to the pollutants increase the experimentally induced dyspnea with antigen aerosols.

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Insulin Allergy

INSULIN PREPARATIONS generally contain mixtures of bovine and porcine insulin which are antigenic in man. Within a few months of the initiation of insulin therapy, circulating antibodies to insulin may be detected. Normally these antibodies are in low concentration, have low insulin-binding capacity and are of no clinical significance. Insulin resistant diabetes is generally associated with high levels of insulin-binding antibodies of the IgG class. IgA, IgM and IgE antibodies may also be found in diabetic sera.

Allergy to insulin has a spectrum of reactions including the frequent local reactions (5 to 30 percent of insulin-treated patients), generalized rashes, serum sickness and anaphylaxis. The skin sensitizing or P-K antibody to insulin is thought to be in the IgE class and is decidedly elevated in patients with anaphylactic sensitivity to insulin. This sensitivity can be partially or completely blocked by production of high levels of IgG blocking antibodies which in turn may lead to insulin resistance. These antibody interactions have many similar features to conventional allergy injection treatment for inhalant allergies and to the protective effect of specific IgG globulin in desensitization in penicillin allergy.

There are a number of steps which may be taken in managing patients with severe insulin allergy: (1) The brand or species of insulin used can be changed; (2) patients may be switched to oral antihyperglycemic agents; (3) insulin with structural modifications such as dealanination, may be used; (4) insulin therapy can be continued with hopes of producing a protective or blocking antibody. This protective effect may later be lost if insulin is discontinued.

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